Response to Baker and Fugh-Berman's Critique of my Paper, "Why has Longevity Increased more in some States than in others?"

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Abstract

Dean Baker and Adriane Fugh-Berman have published a critique of a study I performed in 2007, entitled "Why has longevity increased more in some states than in others?" One of the conclusions I drew from that study was that medical innovation accounts for a substantial portion of recent increases in U.S. life expectancy. Baker and Fugh-Berman claim that my study was subject to a number of major methodological flaws. Many of their claims pertain to the role of infant mortality; the definition of drug vintage; the issue of age adjustment; and the appropriateness of controlling for AIDS, obesity, and smoking in the analysis of longevity. In this article, I make the case that their claims about my study are largely incorrect. I show that infant mortality was not an important determinant of the growth in U.S. life expectancy during the period that I studied, and that my estimates are completely insensitive to the inclusion or exclusion of infant mortality. Controlling for the age distribution of the population also has essentially no effect on the longevity equation estimates. I argue that my definition of drug vintage, based on the initial FDA approval year of a drug's active ingredient, is quite reasonable, and it is consistent with the FDA's evaluation of the therapeutic potential of new drugs. I argue that controlling for AIDS, obesity, and smoking in longevity analysis is entirely appropriate and consistent with the epidemiological literature. Baker and Fugh-Berman express deep skepticism about my study's conclusion that medical innovation has played a very important role in recent U.S. longevity growth, but they offer no explanation of why life expectancy increased by almost a year during 2000-2006, a period of increasing poverty and obesity and declining health insurance coverage.

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Dean Baker and Adriane Fugh-Berman (2009) have published a critique of a study (Lichtenberg (2007)) I performed in 2007, entitled "Why has longevity increased more in some states than in others?" One of the conclusions I drew from that study was that medical innovation accounts for a substantial portion of the recent increase in U.S. life expectancy. Baker and Fugh-Berman claim that my study was subject to a number of major methodological flaws. Many of their claims pertain to the role of infant mortality; the definition of drug vintage; the issue of age adjustment; and the appropriateness of controlling for AIDS, obesity, and smoking in the analysis of longevity. In this article, I will make the case that their claims about my study are largely incorrect.

Infant mortality

Baker and Fugh-Berman make several misleading or incorrect claims related to infant mortality. First, they claimed that my study "fail[ed] to control for infant mortality." However, one of the measures I examined was life expectancy at birth, which depends on the mortality rate of infants as well as the mortality rate of older individuals.[1]

Baker and Fugh-Berman also claim that "infant mortality is the single most important determinant of life expectancy." Infant mortality was not the most important determinant of the growth in U.S. life expectancy during the period that I studied. Data from the National Center for Health Statistics indicate that between 1989-1991 and 2002, life expectancy at birth increased 1.93 years, and life expectancy at age 1 increased by 1.72 years.[2] Only 11% of the increase in life expectancy at birth was due to a reduction

[1] Baker and Fugh-Berman's Figure 1 shows the relationship across states between infant mortality rates and life expectancy at birth in the United States. It is unusual to show a correlation between a variable X and another variable which is calculated using X. Also, the data shown in their Figure 1 (which shows the correlation across states between the *level* of life expectancy and the *level* of the infant mortality rate) are irrelevant to my argument. My study sought to explain interstate variation in the *growth* in life expectancy, not the *level* of life expectancy. Baker and Fugh-Berman fail to distinguish between the level and the growth of life expectancy.

[2]

Life expectancy	1989-1991	2002	change
At birth	75.37	77.3	1.93
At age 1	75.08	76.8	1.72

Source: Arias (2004, Table 11)

in infant mortality.[3] Their claim that "disparities in [infant mortality rates] could account for most differences in longevity in Lichtenberg's analysis" is incorrect.[4]

The sensitivity of my results to infant mortality can easily be assessed by changing the dependent variable in eq. (1) of my study from life expectancy at birth to life expectancy at age 1 or higher ages. The results of this sensitivity analysis are shown in columns 1-3 of Table 1. In column 1 the dependent variable is life expectancy at birth. This is essentially the same equation as the one shown in column 1 of Table 6 of Lichtenberg (2007).[5] In column 2 the dependent variable is life expectancy at age 1, and in column 3 the dependent variable is life expectancy at age 20. The regression coefficients in all three of these equations are almost identical. The estimates are completely insensitive to the inclusion or exclusion of infant mortality.

Measurement of vintage

I defined the vintage of a (single-ingredient) drug as the year in which the drug's active ingredient was first approved by the FDA.[6] All formulations of a drug with the same active ingredient are defined as having the same vintage, even though some formulations may have received FDA approval later than others. Baker and Fugh-Berman seem to think that this way of defining vintage is arbitrary or peculiar. However this way of defining vintage is quite consistent with the FDA's way of classifying new drugs according to their "therapeutic potential."

The FDA classifies all new drug applications as either priority-review or standard-review applications. A *Priority Review* designation is given to drugs that are expected to offer major advances in treatment, or provide a treatment where no adequate

[3] As shown in Table 4 of the MI paper, during the period 1991-2004, the increase in life expectancy at age 65 was 57% as large as the increase in life expectancy at birth

[4] I acknowledged in my paper that "differences in drug vintage explain some of the interstate variation in life expectancy, but the fraction of cross-sectional variance explained is smaller than the fraction of aggregate time-series variance (growth) explained."

[5] The estimates reported in Table 1 allow for clustered standard errors. The estimates reported in Table 6 of Lichtenberg (2007) did not allow for clustering.

[6] The vintage of a combination drug is the *mean* of the years in which the drug's active ingredients were first approved by the FDA.

therapy exists. *Standard Review* is applied to a drug that is expected to offer at most, only minor improvement over existing marketed therapies.[7]

As shown in the following table, based on new drug approvals during the period 1990-2004, new molecular entities were three times as likely to provide significant improvements compared to marketed products in the treatment, diagnosis, or prevention of a disease as new formulations.

	New molecular entities	New formulations
Priority review	183	86
Standard review	248	524
% Priority review	42%	14%

It is therefore quite reasonable, and consistent with FDA evaluation of the therapeutic potential of new drugs, to define the vintage of a drug as the year in which the drug's active ingredient was first approved.

AIDS, obesity, and smoking

The models that I estimated adjusted for AIDS, obesity, and smoking. Baker and Fugh-Berman were critical of this. They argued that this was "an unusual set of variables."

Baker and Fugh-Berman argued that "AIDS is not among the 15 leading causes of death in any state in the U.S., so has a dubious role in this type of analysis." By that standard, infant mortality also has a dubious role: in 1995 (near the peak of the U.S. AIDS epidemic), the number of deaths from HIV/AIDS was 43% larger than the number of infant deaths (42,337 vs. 29,583).

However, that is not a reasonable standard. The effect of a disease on life expectancy depends on the age at which people die from the disease as well as on the frequency of the disease. According to the CDC, the median age at death due to HIV disease in 1995—just before antiretroviral drugs were introduced—was 39 years. Consequently, HIV was the *fourth* largest cause of years of potential life lost (YPLL)

[7] Unfortunately, when a drug is designated a standard-review drug, the FDA does not identify the previously-approved drug(s) to which that drug is considered equivalent.

before age 65 in 1995. The first three were unintentional injuries, cancer, and heart disease. Lichtenberg ( ) provides evidence that pharmaceutical innovation has increased cancer survival rates and reduced age-adjusted cardiovascular mortality rates.

Baker and Fugh-Berman also claimed that "obesity and smoking are risk factors for numerous diseases, but are not acceptable surrogate markers for causes of death." But the CDC has performed studies to estimate mortality attributable to both obesity and smoking. CDC senior epidemiologist Katherine Flegal et al (2005) estimated deaths associated with underweight (body mass index [BMI] <18.5), overweight (BMI 25 to <30), and obesity (BMI ≥30) in the United States in 2000. They found that "underweight and obesity, particularly higher levels of obesity, were associated with increased mortality relative to the normal weight category." CDC (2005) also calculates national estimates of annual smoking-attributable mortality (SAM) and years of potential life lost (YPLL) for adults and infants. It estimates that, during 1997-2001, cigarette smoking and exposure to tobacco smoke resulted in approximately 438,000 premature deaths in the United States and 5.5 million YPLL.

Controlling for population age

Baker and Fugh-Berman claimed that my "analysis [did] not control for age." This claim is not entirely accurate. Also, I will show that controlling for age does not change the basic conclusions.

The most important dependent variable in my study—life expectancy—is based on *age-specific* mortality rates: it controls for (is not affected by) the age distribution of the population.

Drugs in different therapeutic classes tend to be used by different age groups. For example, cardiovascular and cancer drugs tend to be used disproportionately by older people. Some of the models I estimated included a fixed-weight index of drug vintage, rather than a standard (crude) index of drug vintage. The fixed-weight vintage index controls for (holds constant) the distribution of drugs by therapeutic class, which is somewhat correlated with the distribution of drugs by age of user.

The potential bias in my estimates from failure to control for age can be assessed by including a measure of the age distribution in the longevity equation.[8] The equation shown in column 4 of Table 1 includes the fraction of the population that is age 65 or over (old%) as a covariate.[9] The coefficient on this variable is positive but statistically insignificant. Controlling for the age distribution of the population has essentially no effect on the other coefficients.

Income, education, and longevity

Many studies have found a positive correlation between (the level of) socioeconomic status (income or education) and (the level of) life expectancy. Baker and Fugh-Berman argue that the absence of a positive correlation across states between changes in income or education and longevity growth proves that my model is misspecified. However, there is good reason to believe that cross-sectional correlations between longevity and either income or education substantially overestimate the effect of socioeconomic status *per se* on longevity. For example, the positive correlation between income and longevity may reflect the effect of health on income ("reverse causality") as well as the effect of income on health.

Similarly, Almond and Mazumder (2006) argue that, "although it is well known that there is a strong association between education and health, much less is known about how these factors are connected, and whether the relationship is causal." Lleras-Muney (2005) provided perhaps the strongest evidence that education has a causal effect on health. Using state compulsory school laws as instruments, Lleras-Muney found large effects of education on mortality. Almond and Mazumder (2006) revisited these results, noting they are not robust to state time trends, even when the sample is vastly expanded and a coding error rectified. They employed a dataset containing a broad array of health outcomes and found that when using the same instruments, the pattern of effects for specific health conditions appears to depart markedly from prominent theories of how

[8] However, controlling for age in this manner runs the risk of *underestimating* the longevity gains from pharmaceutical innovation. If the causal mechanism is drug vintage ➔ life expectancy ➔ population age, holding population age constant could bias estimates of the effect of drug vintage on life expectancy downward.

[9] The elderly account for about 13% of the U.S. population and about a third of U.S. pharmaceutical use.

education should affect health. They also found suggestive evidence that vaccination against smallpox for school age children may account for some of the improvement in health and its association with education. This raised concerns about using compulsory schooling laws to identify the causal effects of education on health.

Where do longevity gains come from?

Baker and Fugh-Berman express deep skepticism about my study's conclusion that medical innovation has played a very important role in recent U.S. longevity growth. How, then, would they account for the U.S. experience during the period 2000-2006? During that period, the poverty rate increased from 11.3% to 12.3%, median real household income declined about 2%, the share of Americans without health insurance increased from 13.7% to 15.8%, the fraction of Americans who were overweight or obese increased from 56.9% to 61.8%[10]--and life expectancy at birth increased by 0.9 years, from 76.8 to 77.7.[11]

Educational attainment also increased:[12] the fraction of adults who had attended at least some college increased from 51.0% in 2000 to 53.7% in 2006.[13] But the most recent (and largest) estimates of education-related longevity differences, which for reasons discussed above are likely to overstate the effect of education on longevity, imply that increased educational attainment would have increased U.S. life expectancy by only 0.10-0.19 years during the period 2000-2006.[14] Baker and Fugh-Berman offer no explanation of why life expectancy increased so much more than that, during a period of increasing poverty and obesity and declining health insurance coverage.

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[10] Source: Bernstein

[11] Source: Heron

[12] There was also a modest decline in smoking during that period: the fraction of adults who smoked was 22.7% in 1995, 23.2% in 2000, and 20.1% in 2006. Source: BRFSS.

[13] Source: Table A-1. Years of School Completed by People 25 Years and Over, by Age and Sex: Selected Years 1940 to 2008

[14] Meara et al (2008) report that data from the National Longitudinal Mortality Study imply that, during the period 1991-1998, life expectancy at age 25 of people with any college education was 3.7 years higher than that of people with no college education. Data from Multiple Cause of Death files and census data imply that, in the year 2000, life expectancy at age 25 of people with any college education was 7.0 years higher than that of people with no college education

Summary

Dean Baker and Adriane Fugh-Berman have published a critique of a study I performed in 2007, entitled "Why has longevity increased more in some states than in others?" One of the conclusions I drew from that study was that medical innovation accounts for a substantial portion of recent increases in U.S. life expectancy. Baker and Fugh-Berman claim that my study was subject to a number of major methodological flaws. Many of their claims pertain to the role of infant mortality; the definition of drug vintage; the issue of age adjustment; and the appropriateness of controlling for AIDS, obesity, and smoking in the analysis of longevity. In this article, I made the case that their claims about my study are largely incorrect.[15]

I showed that infant mortality was not an important determinant of the growth in U.S. life expectancy during the period that I studied, and that my estimates are completely insensitive to the inclusion or exclusion of infant mortality. Controlling for the age distribution of the population also has essentially no effect on the longevity equation estimates.

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I argued that controlling for AIDS, obesity, and smoking in longevity analysis is entirely appropriate and consistent with the epidemiological literature.

Baker and Fugh-Berman express deep skepticism about my study's conclusion that medical innovation has played a very important role in recent U.S. longevity growth, but they offer no explanation of why life expectancy increased by almost a year during 2000-2006, a period of increasing poverty and obesity and declining health insurance coverage.

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[15] Their article contains other incorrect statements. I did *not* use 2004 payment information from state Medicaid programs and Medicare to calculate the rate of adoption of new prescription drugs in each state. Also, almost all of my studies have appeared or are forthcoming in peer-reviewed journals and books, not only as working papers.

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Table 1
WLS Estimates of Equation 1 Based on the Standard Index of Medicaid Drug Vintage

Column	1	2	3	4
Dependent variable	Life expectancy at birth	Life expectancy at age 1	Life expectancy at age 20	Life expectancy at birth
Independent variables				
vint_medicaid_rx	0.211	0.202	0.203	0.207
Z	5.720	5.602	5.781	5.578
ProbZ	0.000	0.000	0.000	0.000
vint_medicare_rx	0.038	0.038	0.036	0.035
Z	2.258	2.289	2.212	2.057
ProbZ	0.024	0.022	0.027	0.040
aids	-0.026	-0.025	-0.024	-0.026
Z	-8.477	-8.713	-8.067	-9.499
ProbZ	0.000	0.000	0.000	0.000
bmi_gt25	-3.678	-3.446	-3.370	-3.732
Z	-2.089	-2.094	-2.114	-2.137
ProbZ	0.037	0.036	0.034	0.033
now_smoke	-2.149	-2.369	-2.507	-2.198
Z	-1.253	-1.444	-1.613	-1.265
ProbZ	0.210	0.149	0.107	0.206
edu	0.026	-0.042	0.001	0.020
Z	0.097	-0.158	0.005	0.077
ProbZ	0.923	0.874	0.996	0.939
health_cov	0.461	0.403	0.284	0.315
Z	0.372	0.350	0.251	0.252
ProbZ	0.710	0.726	0.802	0.801
income	-1.346	-1.410	-1.174	-1.220
Z	-1.169	-1.297	-1.045	-1.055
ProbZ	0.242	0.194	0.296	0.291
old%				12.987
Z				1.437
ProbZ				0.151

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